

PB86-134459

Health Effects Assessment for
2,4,5-Trichlorophenol

(U.S.) Environmental Protection Agency
Cincinnati, OH

Sep 84



U.S. Department of Commerce
National Technical Information Service

NTIS

PB86-134459

EPA/540/1-86/034
September 1984

HEALTH EFFECTS ASSESSMENT
FOR 2,4,5-TRICHLOROPHENOL

U.S. Environmental Protection Agency
Office of Research and Development
Office of Health and Environmental Assessment
Environmental Criteria and Assessment Office
Cincinnati, OH 45268

U.S. Environmental Protection Agency
Office of Emergency and Remedial Response
Office of Solid Waste and Emergency Response
Washington, DC 20460

REPRODUCED BY
NATIONAL TECHNICAL
INFORMATION SERVICE
U.S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA. 22161

TECHNICAL REPORT DATA <i>(Please read instructions on the reverse before completing)</i>		
1. REPORT NO. EPA/540/1-86/034	2.	3. RECIPIENT'S ACCESSION NO. PB86 134459/AS
4. TITLE AND SUBTITLE Health Effects Assessment for 2,4,5-Trichlorophenol	5. REPORT DATE September 1984	
	6. PERFORMING ORGANIZATION CODE EPA/600/22	
7. AUTHOR(S)	8. PERFORMING ORGANIZATION REPORT NO.	
9. PERFORMING ORGANIZATION NAME AND ADDRESS Environmental Criteria and Assessment Office Office of Research and Development U.S. Environmental Protection Agency Cincinnati, OH 45268	10. PROGRAM ELEMENT NO.	
	11. CONTRACT/GRANT NO.	
12. SPONSORING AGENCY NAME AND ADDRESS Office of Emergency and Remedial Response Office of Solid Waste and Emergency Response U.S. Environmental Protection Agency Washington, DC 20460	13. TYPE OF REPORT AND PERIOD COVERED	
	14. SPONSORING AGENCY CODE	
15. SUPPLEMENTARY NOTES		
16. ABSTRACT This document represents a brief, quantitatively oriented scientific summary of health effects data. It was developed by the Environmental Criteria and Assessment Office to assist the Office of Emergency and Remedial Response in establishing chemical-specific health-related goals of remedial actions. If applicable, chemical-specific subchronic and chronic toxicity interim acceptable intakes are determined for systemic toxicants, or q_1^* values are determined for carcinogens for both oral and inhalation routes. A subchronic and chronic interim acceptable intake was determined for 2,4,5-trichlorophenol based on oral exposure.		
17. KEY WORDS AND DOCUMENT ANALYSIS		
a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
18. DISTRIBUTION STATEMENT Public	19. SECURITY CLASS (This Report) Unclassified	21. NO. OF PAGES 25
	20. SECURITY CLASS (This page) Unclassified	22. PRICE

DISCLAIMER

This report has been funded wholly or in part by the United States Environmental Protection Agency under Contract No. 68-03-3112 to Syracuse Research Corporation. It has been subject to the Agency's peer and administrative review, and it has been approved for publication as an EPA document. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

PREFACE

This report summarizes and evaluates information relevant to a preliminary interim assessment of adverse health effects associated with 2,4,5-trichlorophenol. All estimates of acceptable intakes and carcinogenic potency presented in this document should be considered as preliminary and reflect limited resources allocated to this project. Pertinent toxicologic and environmental data were located through on-line literature searches of the Chemical Abstracts, TOXLINE, CANCERLINE and the CHEMFATE/DATALOG data bases. The basic literature searched supporting this document is current up to September, 1984. Secondary sources of information have also been relied upon in the preparation of this report and represent large-scale health assessment efforts that entail extensive peer and Agency review. The following Office of Health and Environmental Assessment (OHEA) source has been extensively utilized:

U.S. EPA. 1980a. Ambient Water Quality Criteria for Chlorinated Phenols. Environmental Criteria and Assessment Office, Cincinnati, OH. EPA 440/5-80-032. NTIS PB 81-117434.

The intent in these assessments is to suggest acceptable exposure levels whenever sufficient data were available. Values were not derived or larger uncertainty factors were employed when the variable data were limited in scope tending to generate conservative (i.e., protective) estimates. Nevertheless, the interim values presented reflect the relative degree of hazard associated with exposure or risk to the chemical(s) addressed.

Whenever possible, two categories of values have been estimated for systemic toxicants (toxicants for which cancer is not the endpoint of concern). The first, the AIS or acceptable intake subchronic, is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs during a limited time interval (i.e., for an interval that does not constitute a significant portion of the lifespan). This type of exposure estimate has not been extensively used or rigorously defined, as previous risk assessment efforts have been primarily directed towards exposures from toxicants in ambient air or water where lifetime exposure is assumed. Animal data used for AIS estimates generally include exposures with durations of 30-90 days. Subchronic human data are rarely available. Reported exposures are usually from chronic occupational exposure situations or from reports of acute accidental exposure.

The AIC, acceptable intake chronic, is similar in concept to the ADI (acceptable daily intake). It is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs for a significant portion of the lifespan [see U.S. EPA (1980b) for a discussion of this concept]. The AIC is route specific and estimates acceptable exposure for a given route with the implicit assumption that exposure by other routes is insignificant.

Composite scores (CSs) for noncarcinogens have also been calculated where data permitted. These values are used for ranking reportable quantities; the methodology for their development is explained in U.S. EPA (1983a).

For compounds for which there is sufficient evidence of carcinogenicity, AIS and AIC values are not derived. For a discussion of risk assessment methodology for carcinogens refer to U.S. EPA (1980b). Since cancer is a process that is not characterized by a threshold, any exposure contributes an increment of risk. Consequently, derivation of AIS and AIC values would be inappropriate. For carcinogens, q1*s have been computed based on oral and inhalation data if available.

ABSTRACT

In order to place the risk assessment evaluation in proper context, refer to the preface of this document. The preface outlines limitations applicable to all documents of this series as well as the appropriate interpretation and use of the quantitative estimates presented.

A number of subchronic oral studies have been conducted by the same group of investigators. The longest treatment period was 98 days and involved dietary administration of 2,4,5-trichlorophenol to rats. Based on this study an AIS of 70 mg/day and an AIC of 7.0 mg/day were estimated. These estimates, especially the AIC, should be reviewed when additional data are available. A CS of 13 was calculated based on liver and kidney degeneration observed in rats fed a diet containing 3000 ppm in the 98-day dietary study by McCollister et al. (1961).

Pertinent data regarding the potential toxicological effects of inhaled 2,4,5-trichlorophenol could not be located in the available literature.

ACKNOWLEDGEMENTS

The initial draft of this report was prepared by Syracuse Research Corporation under Contract No. 68-03-3112 for EPA's Environmental Criteria and Assessment Office, Cincinnati, OH. Dr. Christopher DeRosa and Karen Blackburn were the Technical Project Monitors and Helen Ball was the Project Officer. The final documents in this series were prepared for the Office of Emergency and Remedial Response, Washington, DC.

Scientists from the following U.S. EPA offices provided review comments for this document series:

Environmental Criteria and Assessment Office, Cincinnati, OH
Carcinogen Assessment Group
Office of Air Quality Planning and Standards
Office of Solid Waste
Office of Toxic Substances
Office of Drinking Water

Editorial review for the document series was provided by:

Judith Olsen and Erma Durden
Environmental Criteria and Assessment Office
Cincinnati, OH

Technical support services for the document series was provided by:

Bette Zwayer, Pat Daunt, Karen Mann and Jacky Bohanon
Environmental Criteria and Assessment Office
Cincinnati, OH

TABLE OF CONTENTS

	<u>Page</u>
1. ENVIRONMENTAL CHEMISTRY AND FATE.	1
2. ABSORPTION FACTORS IN HUMANS AND EXPERIMENTAL ANIMALS	3
2.1. ORAL	3
2.2. INHALATION	3
3. TOXICITY IN HUMANS AND EXPERIMENTAL ANIMALS	4
3.1. SUBCHRONIC	4
3.1.1. Oral.	4
3.1.2. Inhalation.	5
3.2. CHRONIC.	5
3.2.1. Oral.	5
3.2.2. Inhalation.	5
3.3. TERATOGENICITY AND OTHER REPRODUCTIVE EFFECTS.	5
3.3.1. Oral.	5
3.3.2. Inhalation.	5
3.4. TOXICANT INTERACTIONS.	5
4. CARCINOGENICITY	6
4.1. HUMAN DATA	6
4.1.1. Oral.	6
4.1.2. Inhalation.	6
4.2. BIOASSAYS.	6
4.2.1. Oral.	6
4.2.2. Inhalation.	6
4.3. OTHER RELEVANT DATA.	6
4.4. WEIGHT OF EVIDENCE	6
5. REGULATORY STANDARDS AND CRITERIA	8

TABLE OF CONTENTS (cont.)

	<u>Page</u>
6. RISK ASSESSMENT	9
6.1. ACCEPTABLE INTAKE SUBCHRONIC (AIS)	9
6.1.1. Oral	9
6.1.2. Inhalation	9
6.2. ACCEPTABLE INTAKE CHRONIC (AIC).	9
6.2.1. Oral	9
6.2.2. Inhalation	10
6.3. CARCINOGENIC POTENCY (q ₁ *)	10
8. REFERENCES.	11
APPENDIX: Summary Table for 2,4,5-Trichlorophenol.	14

LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
AIC	Acceptable intake chronic
AIS	Acceptable intake subchronic
BCF	Bioconcentration factor
bw	Body weight
CAS	Chemical Abstract Service
CS	Composite score
DMBA	Dimethybenz[a]anthracene
LOAEL	Lowest-observed-adverse-effect level
MED	Minimum effective dose
NOEL	No-observed-effect level
ppm	Parts per million
RV _d	Dose-rating value
RV _e	Effect-rating value
UF	Uncertainty factor

1. ENVIRONMENTAL CHEMISTRY AND FATE

The relevant physical and chemical properties and environmental fate of 2,4,5-trichlorophenol (CAS No. 95-95-4) are given in Table 1-1.

The half-life values for 2,4,5-trichlorophenol in air and aquatic media were not located in the available literature; however, based on the speculated fate of its isomer (see accompanying report on 2,4,6-trichlorophenol), photodecomposition in the atmosphere is expected to be the dominant fate of this compound. Similarly, photodecomposition is expected to play a significant role in determining the fate of this chemical in aquatic media since this compound may be resistant to microbial degradation (U.S. EPA, 1980a). In the absence of quantitative data pertaining to the rate of photodecomposition, it is not possible to predict the half-lives of this chemical in air and aquatic media.

The octanol/water partition coefficient value given in Table 1-1 indicates that this compound may be significantly sorbed to soils with high organic carbon content. In sandy soils, 2,4,5-trichlorophenol may have significant mobility. The biodegradability and aqueous solubility of this compound are such that leaching of this compound, especially from sandy soil to groundwater, is possible.

TABLE 1-1
Selected Physical and Chemical Properties and
Half-Lives for 2,4,5-Trichlorophenol

Properties	Values	References
Chemical class:	halogenated phenol	NA
Vapor pressure:	1 mm at 72°C	Weast, 1980
Water solubility:	1190 mg/kg at 25°C	Verschueren, 1983
Log octanol/water partition coefficient:	3.72	U.S. EPA, 1980a
BCF:	290	Veith et al., 1979
	110	U.S. EPA, 1980a
Residence time in soil:	>72 days for complete disappearance	Verschueren, 1983

NA = Not applicable

2. ABSORPTION FACTORS IN HUMANS AND EXPERIMENTAL ANIMALS

2.1. ORAL

No quantitative studies of absorption following oral exposure of humans or animals to 2,4,5-trichlorophenol could be located in the available literature. Dougherty and Piotrowska (1976) suggested that chlorophenols as a class tend to be eliminated rapidly in the urine; therefore, urinary concentrations reflect exposure. Quantitative analyses were not performed in this study.

Kutz et al. (1978) analyzed 418 samples of urine collected from the general population as part of a health and nutritional survey. Residues of 2,4,5-trichlorophenol were found in 1.7% of the samples. The average concentration found was $<5 \mu\text{g/l}$ and the maximum was $32.4 \mu\text{g/l}$.

2.2. INHALATION

Pertinent data regarding the absorption of 2,4,5-trichlorophenol following inhalation exposure could not be located in the available literature.

3. TOXICITY IN HUMANS AND EXPERIMENTAL ANIMALS

3.1. SUBCHRONIC

3.1.1. Oral. McCollister et al. (1961) conducted a variety of toxicity studies on 2,4,5-trichlorophenol in rats and rabbits. Groups of rabbits (number and sex unspecified) were given 20 daily oral (gavage) doses of 1, 10, 100 or 500 mg 2,4,5-trichlorophenol/kg bw over a 28-day period. "Very slight kidney changes" and "very slight liver and kidney changes" were found in one rabbit each from the 100 mg/kg and 500 mg/kg groups, respectively. No effects were found in five rabbits given 1 or 10 mg/kg daily.

Concurrently, McCollister et al. (1961) administered 18 daily doses of 0, 30, 100, 300 or 1000 mg 2,4,5-trichlorophenol/kg bw in olive oil to groups of five male rats over a 24-day period. Rats treated with 1000 mg/kg bw experienced a small amount of weight loss (~10 g; initial body weight ~270 g) during the first 10 days, but recovered quickly and were gaining weight normally by day 14. Upon necropsy, high dose rats were found to have slight kidney enlargement when compared to control rats. No other effects of treatment were found; in this study, 300 mg 2,4,5-trichlorophenol/kg bw was a NOEL in rats.

McCollister et al. (1961) also maintained groups of 10 male and 10 female rats on diets containing 100, 300, 1000, 3000 or 10,000 ppm 2,4,5-trichlorophenol for 98 days. Mild diuresis and slight degenerative changes in the liver and kidneys were noted in rats of both sexes in the 3000 and 10,000 ppm groups. Reported pathological lesions in the liver consisted of mild centrilobular degenerative changes characterized by cloudy swelling and occasional areas of focal necrosis. Slight proliferation of the bile ducts and early portal cirrhosis were also observed. The kidneys showed moderate

changes in the epithelium of the convoluted tubules and "early proliferation" of the interstitial tissue. These changes were considered to be mild and reversible. Judging from behavior, mortality, food consumption, growth, terminal hematology, body and organ weights and gross and microscopic pathology, 1000 ppm was considered to be a NOEL.

No studies of subchronic oral exposure of humans to 2,4,5-trichlorophenol could be located in the available literature.

3.1.2. Inhalation. No reports of toxicity in humans or animals associated with inhalation exposure to 2,4,5-trichlorophenol could be located in the available literature.

3.2. CHRONIC

3.2.1. Oral. No studies of toxicity associated with chronic oral exposure to 2,4,5-trichlorophenol in humans or animals could be located in the available literature.

3.2.2. Inhalation. No studies of toxicity associated with chronic inhalation exposure to 2,4,5-trichlorophenol could be located in the available literature.

3.3. TERATOGENICITY AND OTHER REPRODUCTIVE EFFECTS

3.3.1. Oral. No reports of teratogenicity in humans or animals associated with oral exposure to 2,4,5-trichlorophenol could be located in the available literature.

3.3.2. Inhalation. No reports of teratogenicity in humans or animals associated with inhalation exposure to 2,4,5-trichlorophenol could be located in the available literature.

3.4. TOXICANT INTERACTIONS

No studies of the interactions of 2,4,5-trichlorophenol with other xenobiotics could be located in the available literature.

4. CARCINOGENICITY

4.1. HUMAN DATA

4.1.1. Oral. No reports of carcinogenicity in humans associated with oral exposure to 2,4,5-trichlorophenol could be located in the available literature.

4.1.2. Inhalation. No reports of carcinogenicity in humans associated with inhalation exposure to 2,4,5-trichlorophenol could be located in the available literature.

4.2. BIOASSAYS

4.2.1. Oral. No studies of the carcinogenicity of orally administered 2,4,5-trichlorophenol could be located in the available literature.

4.2.2. Inhalation. No studies of the carcinogenicity of 2,4,5-trichlorophenol administered by inhalation could be located in the available literature.

4.3. OTHER RELEVANT DATA

Rasanen et al. (1977) tested 2,4,5-trichlorophenol for mutagenicity in the Ames assay using Salmonella typhimurium strains TA98, TA100, TA1535 and TA1537. Negative results were reported both in the presence and absence of rat hepatic S9 fraction. Details of protocol were not available.

Repeated spraying of the flower buds of vetch (Vicia faba) with an aqueous solution of 2,4,5-trichlorophenol increased the frequency of abnormalities in pollen mother cells, including stickiness and chromosomal lagging during cell division and an increased incidence in fragmented chromosomes (Amer and Ali, 1974).

4.4. WEIGHT OF EVIDENCE

No reports of cancer in humans associated with 2,4,5-trichlorophenol and no bioassays of the carcinogenicity of 2,4,5-trichlorophenol in laboratory animals could be located in the available literature.

Boutwell and Bosch (1959) treated 20 female Sutter mice with a single application of 75 μ g DMBA on the dorsal skin as a tumor initiator. Approximately 1 drop of commercial grade 2,4,5-trichlorophenol (~25 μ l) in acetone was applied twice weekly for 16 weeks as a tumor promotor. Skin papillomas were observed in 8/19 surviving mice. No skin tumors were found in 18 surviving controls treated with acetone alone, and 1 tumor was found in 21 surviving male mice (strain unspecified) treated with 75 μ g DMBA alone. No mice were treated with 2,4,5-trichlorophenol alone.

Applying the criteria proposed by the Carcinogen Assessment Group of the U.S. EPA for evaluating the overall weight of evidence of carcinogenicity for humans (Federal Register, 1984), 2,4,5-trichlorophenol is most appropriately classified a Group D - Not Classified chemical.

5. REGULATORY STANDARDS AND CRITERIA

No current standards or criteria for 2,4,5-trichlorophenol in air were located in the available literature.

The U.S. EPA (1980a) calculated an interim ADI of 7 mg 2,4,5-trichlorophenol/day for a 70 kg human, based on the NOEL of 1000 ppm from the 98-day oral study in rats by McCollister et al. (1961), assuming that rats eat food equivalent to 10% of their body weight/day and applying a UF of 1000. Assuming that humans drink 2 l of water/day and eat 6.5 g of fish, which bioaccumulate 2,4,5-trichlorophenol by a factor of 110 (U.S. EPA, 1980a), an ambient water quality criterion of 2.6 mg/l was determined by the U.S. EPA (1980a). If human exposure is assumed to be from the consumption of fish and shellfish alone, an ambient water quality criterion of 9.8 mg/l has been determined to be protective. On the basis of organoleptic effects, a criterion of 1.0 µg/l has been suggested.

6. RISK ASSESSMENT

6.1. ACCEPTABLE INTAKE SUBCHRONIC (AIS)

6.1.1. Oral. McCollister et al. (1961) exposed rats of each sex to diets containing 100, 300, 1000, 3000 or 10,000 ppm 2,4,5-trichlorophenol for 98 days. Mild pathological changes in the kidneys and liver were observed in rats of both sexes exposed to diets containing 3000 or 10,000 ppm 2,4,5-trichlorophenol. No effects were observed in rats exposed to 1000 ppm, and this level was judged to be a NOEL. Assuming that young, growing rats consume food equivalent to 10% of their body weight/day, this diet resulted in an animal intake of 100 mg/kg/day. An AIS can be calculated by multiplying the animal dose, 100 mg/kg/day, by 70 kg (the average weight of a man) and dividing by an UF of 100 (a factor of 10 to account for interspecies extrapolation and a factor of 10 to protect unusually sensitive groups in the population). An AIS of 70 mg/day is calculated.

6.1.2. Inhalation. No data concerning subchronic inhalation exposure of humans or animals to 2,4,5-trichlorophenol have been located in the available literature; hence, no AIS for subchronic inhalation exposure can be calculated.

6.2. ACCEPTABLE INTAKE CHRONIC (AIC)

6.2.1. Oral. Data from the study by McCollister et al. (1961), from which an AIS of 70 mg/day in humans was calculated, can be used to derive an AIC for oral exposure, since no studies of truly chronic oral exposure were found. Application of an additional UF of 10 to convert from a subchronic NOEL to a chronic NOEL results in an AIC for oral exposure of 7.0 mg 2,4,5-trichlorophenol/day.

U.S. EPA (1983b) computed a CS for the effects of mild diuresis and slight degenerative changes in the liver and kidneys observed by McCollister

6. RISK ASSESSMENT

6.1. ACCEPTABLE INTAKE SUBCHRONIC (AIS)

6.1.1. Oral. McCollister et al. (1961) exposed rats of each sex to diets containing 100, 300, 1000, 3000 or 10,000 ppm 2,4,5-trichlorophenol for 98 days. Mild pathological changes in the kidneys and liver were observed in rats of both sexes exposed to diets containing 3000 or 10,000 ppm 2,4,5-trichlorophenol. No effects were observed in rats exposed to 1000 ppm, and this level was judged to be a NOEL. Assuming that young, growing rats consume food equivalent to 10% of their body weight/day, this diet resulted in an animal intake of 100 mg/kg/day. An AIS can be calculated by multiplying the animal dose, 100 mg/kg/day, by 70 kg (the average weight of a man) and dividing by an UF of 100 (a factor of 10 to account for interspecies extrapolation and a factor of 10 to protect unusually sensitive groups in the population). An AIS of 70 mg/day is calculated.

6.1.2. Inhalation. No data concerning subchronic inhalation exposure of humans or animals to 2,4,5-trichlorophenol have been located in the available literature; hence, no AIS for subchronic inhalation exposure can be calculated.

6.2. ACCEPTABLE INTAKE CHRONIC (AIC)

6.2.1. Oral. Data from the study by McCollister et al. (1961), from which an AIS of 70 mg/day in humans was calculated, can be used to derive an AIC for oral exposure, since no studies of truly chronic oral exposure were found. Application of an additional UF of 10 to convert from a subchronic NOEL to a chronic NOEL results in an AIC for oral exposure of 7.0 mg 2,4,5-trichlorophenol/day.

U.S. EPA (1983b) computed a CS for the effects of mild diuresis and slight degenerative changes in the liver and kidneys observed by McCollister

et al. (1961) in rats fed diets containing 3000 ppm 2,4,5-trichlorophenol. Assuming rats eat food equivalent to 5% of their body weight/day, the effective animal dose is 150 mg/kg/day. The equivalent human MED, 179.6 mg/day, is calculated by multiplying the animal dose by the cube root of the ratio of the body weight of rats (assumed: 0.35 kg) to that of humans (assumed: 70 kg), applying an UF of 10 to convert from subchronic to chronic exposure, and multiplying the result by 70 to express the dose as mg/day for a 70 kg human. This MED corresponds to an RV_d of 2.1. The effects observed, mild diuresis and degenerative changes in the liver and kidney, warrant an RV_e of 6. A CS of 13, the product of RV_d and RV_e , results.

6.2.2. Inhalation. No reports of toxicity in humans and animals associated with chronic inhalation exposure to 2,4,5-trichlorophenol could be located in the available literature; hence, no AIC for chronic inhalation exposure can be calculated.

6.3. CARCINOGENIC POTENCY (q_1^*)

No reports of carcinogenicity in humans or animals exposed to 2,4,5-trichlorophenol by either the oral or the inhalation route could be located in the available literature. No carcinogenic potency estimate can be made for either oral or inhalation exposure to 2,4,5-trichlorophenol.

8. REFERENCES

Amer, S.M. and E.M. Ali. 1974. Cytologic effects of pesticides. V. Effects of some herbicides on Specia faba. Cytologia. 33: 633. (Cited in NIOSH, 1981)

Boutwell, R.K. and D.K. Bosch. 1959. The tumor-promoting action of phenol and related compounds for mouse skin. Cancer Res. 19: 413-424. (Cited in U.S. EPA, 1980a)

Dougherty, R.C. and K. Piotrowska. 1976. Screening by negative chemical ionization mass spectrometry for environmental contamination with toxic residues: Application to human urines. Proc. Natl. Acad. Sci., USA. 73: 1777-1781. (Cited in U.S. EPA, 1980a)

Federal Register. 1984. Environmental Protection Agency. Proposed guidelines for carcinogenic risk assessment. 49 FR 46294-46299.

Kutz, F.W., R.S. Murphy and S.C. Strassman. 1978. Survey of Pesticide Residues and Their Metabolites in Urine from the General Population. In: Pentochlorophenol: Chemistry, Pharmacology and Environmental Toxicology, K.R. Rao, Ed., Plenum Press, NY. p. 363-369. (Cited in U.S. EPA, 1980a)

McCollister, D.D., D.T. Lockwood and V.K. Rowe. 1961. Toxicologic information on 2,4,5-trichlorophenol. Toxicol. Appl. Pharmacol. 3: 63-70. (Cited in U.S. EPA, 1980a)

NIOSH (National Institute for Occupational Safety and Health). 1981. Information Profiles on Potential Occupational Hazards. Chlorophenols. Second Draft. Prepared by Syracuse Research Corporation under Contract No. 21-79-0030 for NIOSH, Rockville, MD.

Rasanen, L., M.L. Hattula and A.U. Arstila. 1977. The mutagenicity of MCPA and its soil metabolites, chlorinated phenols, catechols and some widely used slimicides in Finland. Bull. Environ. Contam. Toxicol. 18: 565-571. (Cited in U.S. EPA, 1980a)

U.S. EPA. 1980a. Ambient Water Quality Criteria for Chlorinated Phenols. Environmental Criteria and Assessment Office, Cincinnati, OH. EPA 440/5-80-032. NTIS PB 81-117434.

U.S. EPA. 1980b. Guidelines and Methodology Used in the Preparation of Health Effects Assessment Chapters of the Consent Decree Water Quality Criteria. Federal Register. 45:79347-79357.

U.S. EPA. 1983a. Methodology and Guidelines for Reportable Quantity Determinations Based on Chronic Toxicity Data. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, OHEA for the Office of Solid Waste and Emergency Response, Washington, DC.

U.S. EPA. 1983b. Reportable Quantity for 2,4,5-Trichlorophenol. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, OHEA for the Office of Solid Waste and Emergency Response, Washington, DC.

Veith, G.D., D.L. Defoe and B.V. Bergstedt. 1979. Measuring and estimating the bioconcentration factor of chemicals in fish. J. Fish Res. Board Can. 36: 1040-1048.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemistry, 2nd ed. Van Nostrand Reinhold Co., NY. p. 1310.

Weast, R.C., Ed. 1980. CRC Handbook of Chemistry and Physics. CRC Press, Inc., Boca Raton, FL. p. D207.

APPENDIX

Summary Table for 2,4,5-Trichlorophenol

Species	Experimental Dose/Exposure	Effect	Acceptable Intake (AIS or AIC)	Reference
Inhalation				
AIS			ND	
AIC			ND	
Oral				
AIS	rat 1000 ppm diet (100 mg/kg bw/day) ^a	none	70 mg/day	McCollister et al., 1961
AIC	rat 1000 ppm diet (100 mg/kg bw/day) ^a	none	7.0 mg/day	McCollister et al., 1961
Maximum composite score	rat 3000 ppm diet (150 mg/kg bw/day) ^b (RV _d = 2.1) ^c	mild diuresis, liver and kidney degeneration (RV _e = 6)	13	McCollister et al., 1961; U.S. EPA, 1983b

^afood factor of 10% bw assumed for young growing rats

^bfood factor of 5% bw was assumed for rats

CUF of 10 was applied to convert from subchronic to chronic exposures

ND = Not derived